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" Scire est nescire, nisi id me
Scire alius sciret."

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THE ATOMIC BOMB*

I: THE RELEASE OF ATOMIC ENERGY AND THE PATHOLOGICAL EFFECTS OF RADIATION

BY

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An outline of the scientific background behind the explosive release of atomic energy should be not only of general interest but also of practical assistance. It should help doctors to anticipate the effects of the atomic bomb in circumstances not exactly covered by the practical account, or to follow accounts of new ideas yet to be published.

The atoms of the various chemical elements are each composed of an atomic nucleus in the centre, and one or more electrons circulating round the nucleus like the moon round the earth. The nucleus is very small and relatively heavy. The electrons are very light and each one carries one negative charge of electricity. The nuclei of atoms are always built up of two kinds of particles, protons and neutrons, in different proportions.

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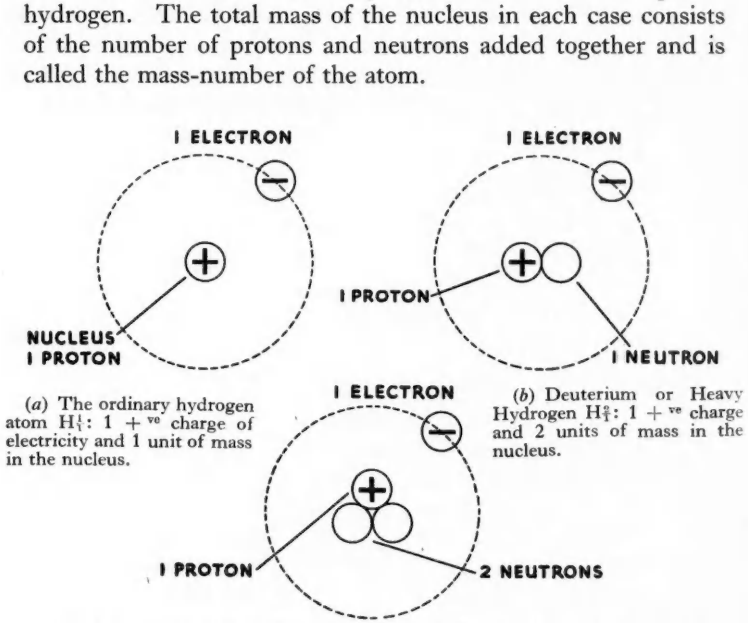
Protons are atomic particles which carry one positive charge of electricity and have a mass of one unit. Neutrons have a mass of one unit but no electric charge.

Figure 1 illustrates this by showing three kinds of hydrogen atoms. The variety of an atom is determined by the number of protons (*i.e.* positive charges) in its nucleus. So all these atoms are hydrogen, because they all have only one proton in the nucleus, one proton being characteristic of hydrogen. But these three atoms are different, because they have different numbers of neutrons: and they are called different isotopes of hydrogen. The total mass of the nucleus in each case consists of the number of protons and neutrons added together and is called the mass-number of the atom.



(*a*) The ordinary hydrogen atom H_1^1: 1 + ve charge of electricity and 1 unit of mass in the nucleus.

(*b*) Deuterium or Heavy Hydrogen H_1^2: 1 + ve charge and 2 units of mass in the nucleus.

(*c*) Tritium H_1^3 (radioactive): 1 + ve charge and 3 units of mass in the nucleus.

FIGURE 1

Radioactivity. As well as illustrating the occurrence of isotopes, figure 1 illustrates the occurrence of radioactivity. The nuclei of atoms are not composed of protons and neutrons gathered together in any proportions: only a few of the many possible combinations of protons and neutrons will in fact stick

together. In the lighter elements about equal numbers of each are needed to form a stable nucleus, and in the heavier atoms about two protons for every three neutrons. An atom of *Tritium* or H^3, which has two neutrons to only one proton, tends to fly apart, *i.e.* it is radioactive (Figure 1).

The heaviest atom which is *stable* is that of Bismuth with 209 protons and neutrons in the nucleus, *i.e.* B^{209}. All atoms heavier than this are unstable or in other words radioactive: for example, Ur^{235}, Ur^{238} and Ur^{239}, the three natural isotopes of uranium, which is the heaviest natural element. During ordinary radioactivity the nucleus of an unstable atom does not split into two halves. It merely keeps on losing small particles until it reaches a stable condition. The types of particles it emits are:

1. *Alpha particles.* These have a mass of four, and two positive charges on each.

2. *Beta particles*, with a very small mass and one negative charge. These are accompanied by

3. *Gamma rays*, which are most important and responsible for most of the radiation casualties from the atomic bomb.

Atomic Fission

There is such a thing, however, as a splitting of the atoms. This is Atomic Fission, discovered in Berlin in 1939 by Hahn and Strassmann. There are everywhere a few stray neutrons moving about. If a Ur^{235} atom is struck by one stray neutron it absorbs this neutron and becomes for an instant Ur^{236}. The Ur^{236} atom cannot exist for more than an instant however. It splits down the middle and this produces: (*a*) two smaller atoms, *e.g.* Barium and Krypton (or many other pairs); (*b*) three or four spare neutrons, and (*c*) very much energy. If a mass of Ur^{235} atoms is present this effect is propagated, due to the spare neutrons setting off more Ur atoms and so on. This is the "chain reaction": within a fraction of a second it has built up into:

The Atomic Explosion. Most of the fission products are not natural elements and so are extremely radioactive. They produce rays which are very intense for about ten seconds. But unless the Ur^{235} is pure this explosion will not happen because

the impurities waste all the neutrons. To make an atom bomb, therefore, all that is needed is to collect a large enough mass of pure Ur^{235}—a very difficult thing—and to bring the mass together only when an explosion is needed, since a mass smaller than a certain critical size will not explode. Ur^{235} occurs as only 0·7 per cent. of natural uranium, most of which is Ur^{238}.

In practice, another element, plutonium made from Ur^{238} in the atomic pile is now used. The great energy of the bomb is due to the fact that during atomic fission some of the mass of the uranium is converted into energy.

This is shown by the formula: $E = MC^2$

Where E = energy formed; M = the mass lost; C = the velocity of light $= 3 \times 10^{10}$ cms./sec., so that $C^2 = 9 \times 10^{20}$, a very big number: and the energy E is enormous.

The Decay of Radioactive Material

As the atoms of any radioactive material are constantly breaking down, it must be steadily disappearing. This is illustrated by figure 2 which is the decay curve for radium. It shows that in 1,690 years the radium one starts with has decayed to half. At the end of another 1,690 years, one-half of this remaining half has gone, *i.e.* one-quarter remains. At the end of a third 1,690 years only one-eighth and so on, but the radium never entirely disappears. This is expressed by saying that its "half-life" is 1,690 years (Figure 2).

Fortunately the half-life of the fission products of the bomb is mostly very much shorter, and is measured in seconds rather than years. But some long-lived elements are formed which fade away very slowly until negligible amounts are left.

We must now consider the properties of the gamma rays emitted by the fission products during and after the atomic explosion. Alpha, beta and neutron rays will be ignored now as they do not in practice produce many casualties. The properties of gamma rays which matter to us are:

1. Penetration.
2. "Half-value-layer."
3. Straight line path—inverse square law.
4. Ionization.

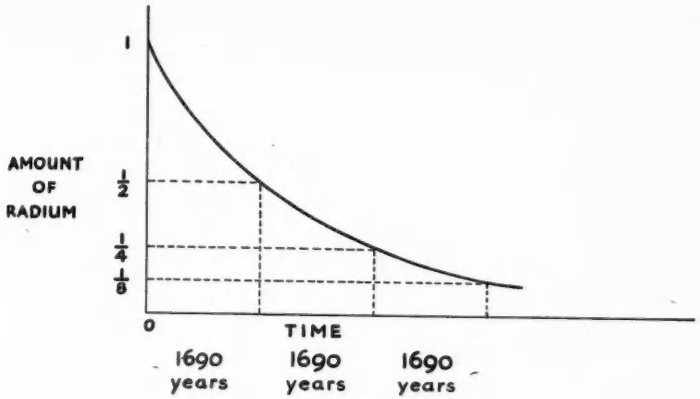


FIGURE 2
Decay curve for radium

Penetration. The gamma rays from the bomb have very great powers of penetration. They traverse $3\frac{1}{2}$ inches of concrete before they are reduced to half-strength. Alternatively 0·6 inches of lead, 1 inch of steel or 8 inches of water all reduce them to half-strength. This means that people within buildings are only very incompletely protected from gamma rays.

"*Half-Value-Layer*". The strength of gamma rays falls after penetrating an increasing thickness of concrete. The first $3\frac{1}{2}$ inches traversed reduces the gamma rays to half the original strength, the next $3\frac{1}{2}$ inches reduces this half to half again, *i.e.* to one-quarter the original, the next $3\frac{1}{2}$ inches reduces the rays to one-eighth and so on: but no thickness will cut them out entirely. The thickness of any substance needed to cut the gamma rays to half is called the "half-value-layer" of the substance: *e.g.* $3\frac{1}{2}$ inches of concrete or 1 inch of steel. All this means that considerable thicknesses are needed to shield effectively against the rays.

Straight Line Path. Gamma rays are similar to light rays: they only travel in straight lines. This has one important effect. The beam of rays to which an individual is subjected will broaden out like the beam from a headlamp. So the area covered by the beam of rays increases with the square of the distance from the source, and the intensity of radiation varies

inversely as the square of the distance from the bomb. Therefore, as a protective factor a little extra distance is worth a lot of screening material. This principle applies, of course, to hospital radium as much as to the bomb.

Ionization. As stated above and illustrated in figure 1, all atoms are encircled by one or more electrons which travel round in an orbit. Normally the number of electrons around the outside of an atom must equal the number of protons (positive electric charges) in the nucleus. However, a gamma ray may strike an atom and knock off one of these electrons. This leaves an unbalanced positive charge on the nucleus and now the atom is *ionized.*

The unit of dose of gamma rays received by an individual is called the Roentgen, signified by the symbol " r ", and it is measured by the ionization it produces under standard conditions. The effect of so many r falling on an individual depends not only on the number of r on each square centimetre, but also on the size of the part irradiated. Exposure to radiation from the atom bomb, however, nearly always involves the whole body: the effects of dosage will be given later on that assumption.

Ionization is also responsible for the biological effects of radiation. The molecules of body substances are changed by it. The effects appear to be purely destructive, and there is no evidence of a stimulating effect. The effects of radiation are, however, selective, for the sensitivity of different tissues to ionization varies. For example, in the treatment of radiosensitive growths the normal cells are less easily killed than the malignant. And in the case of irradiation of whole animals by the atomic bomb or by experiment the effects are still selective: some tissues are more sensitive than others, also different animals differ in their sensitivity.

For example, for gamma rays:
200r kills 50 per cent. of guinea pigs.
500r kills 50 per cent. of men.*
800r kills 50 per cent. of rabbits.

The tissues themselves are affected in the following order of sensitivity which is similar for all animals: lymphatic tissue,

* This is the maximum of a suggested range of 300–500r based on observations in Japan.

germinal epithelium of testis, bone marrow, gastrointestinal, ovary, skin, connective tissue, bone, liver, pancreas, kidney, nerve, brain, muscle.

We will deal below with the first few of these. Effects on the others are not seen in whole-body-irradiation, as the individual dies from the effects on the sensitive tissues before the resistant can be affected. The fatal effects of radiation are threshold effects: less than a certain dose per day or week will have no effect however long continued. The threshold varies for different tissues. Recovery from radiation damage does occur but it is not always complete. It is thought that up to a tolerance dose of 0·5r per week can be accepted without damage by an individual. This is the dose used for calculating safety precautions in hospitals. Non-threshold effects do occur, but are more rare. The most important are the genetic effects due to direct hits by the radiation on the genes. I propose to say nothing about these. Little is known of their importance, and even in the populations of Hiroshima and Nagasaki several generations may be needed for the effects of mutations to appear. As well as direct action by ionization within the cell producing damage there is some evidence for humoral effects involving the production of diffusable toxic substances, both in local effects and the general radiation sickness.

The General Effects of a Single Large Exposure to Gamma Rays

We will now concentrate on the acute radiation effects of the single large dose which comes from the explosion of the bomb. The effects of chronic dosage are concerned with more peaceful hazards, except if there should be an area of ground contamination from the bomb and essential work has to be carried on by people within the area.

There are three groups of fatal cases:

1. Shock may cause death in five to forty-eight hours.

2. Toxaemia may cause death in five to fifteen days (ten to fifteen days mostly), after initial improvement, especially following the mid-lethal dose of 400–500r.

3. Haemopoeitic damage may cause death in thirty to ninety days.

The toxaemic effects are:

(*a*) *Fever.* A step-ladder temperature sometimes going on to hyperpyrexia and death, commencing two to twenty-eight days after exposure. In non-fatal cases this usually starts in the third week, at about the same time as epilation.

(*b*) A Heparin-like substance is produced, causing loss of clotting power and increased bleeding time, an important factor in severe cases dying within seven days.

The more commonly seen haemorrhagic state occurs at three to four weeks and is associated with capillary damage. After the first seven days the commonest cause of death is agranulocytosis, aggravated by the fever and haemorrhages. The effects on the blood forming organs have been most studied and are of most interest. The lymphocytes are the most sensitive of all cells. They reach a minimum, in the blood, in about three to five days and may nearly completely disappear after a heavy exposure. The germinal centres of the lymph nodes disappear, but recovery begins very soon afterwards. The blood-forming elements of the bone-marrow are damaged and in severe cases nearly disappear (except in small islets) within two weeks. Owing to hyperaemia the marrow remains red. Recovery usually begins before four weeks in a non-fatal case. The circulating granulocytes reach a minimum at about six to fourteen days after exposure. The platelets reach their minimum about one week later and the red cells after about twenty-one days. The anaemia may be very slow to recover, taking several months. The sedimentation rate rises after one week.

The Gonads. Germinal epithelium and developing ova are very sensitive and temporary sterility is common. They are, however, replaced after a varying interval in nearly all surviving cases, and the dose of total body radiation needed to cause permanent sterility is more than the fatal dose.

Gastro-Intestinal Epithelium. Oedema sometimes followed by false membrane formation occurs after two to three days in severe cases. Ulceration and submucous haemorrhages are also produced.

The Skin. Erythema is not likely to be seen as it needs a dose well above the lethal dose (up to 1,000r).

Epilation is often seen, and it has been found in Japan that cases which are not epilated usually recover. It occurs towards the end of the third week.

Effects due to the ingestion of radioactive elements may be met with and if they do occur can be most serious, especially when elements, *e.g.* radioactive strontium or plutonium, are deposited in the bones. This may result in bone-marrow damage long afterwards, *i.e.* anaemia and agranulocytosis, or sometimes in leukaemia or osteogenic sarcoma.

THE ATOMIC BOMB
II: THE EFFECTS OF THE EXPLOSION

BY

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The atomic bomb resembles bombs of the more conventional type in so far as the explosive effect is the result of the very rapid liberation of a large quantity of energy in a relatively small space. It differs however from other bombs in three important respects. First, the amount of energy released by an atomic bomb is a thousand or more times as great as that produced by the most powerful T.N.T. bombs. Secondly, the explosion is accompanied by highly penetrating and deleterious invisible rays, in addition to intense heat and light. Finally, the substances remaining after the explosion are radioactive, emitting harmful radiations.

The effects to be described are those that would be produced by the explosion of a " nominal " atomic bomb of the type dropped over Japan. Such a bomb has an effective energy release roughly equivalent to that produced by the explosion of 20,000 tons of T.N.T., and can be exploded in the air, under

water or on the ground. Over this country the air burst is probably the optimum site for detonation, but the possibility of an under-water explosion with consequent widespread contamination by radioactive products carried in water droplets must not be overlooked. The bombs over Japan were exploded in the air at a height of about 2,000 feet. To obtain a comparable effect on a British city in which the buildings on the whole would be more resistant to heat and blast, the explosion would need to occur within a height range of from 500 to 1,000 feet.

When an atomic bomb is detonated in the air the first sign that anything has happened is a brilliant flash of bluish white light lasting a small fraction of a second. The light is accompanied by a flash of radiant heat which lasts a second or two after the intense brilliance is over, and which can ignite combustible materials up to a distance of two miles. At the same time the pressure wave is released and the hot source expands into a ball of fire several hundred feet in diameter. This ball of fire, surrounded for a few seconds by a ring of fog caused by a succeeding negative pressure wave, is then seen rushing upwards, expanding as it goes. It is followed by an opaque cloud of turbulent gases, and reaches the stratosphere in about four minutes. By this time the fire ball has disappeared and has been replaced by a mushroom-shaped cloud of white vapour some two miles in diameter.

The bulk of the radiation, consisting of alpha and beta particles, neutrons and gamma rays is emitted during the first second after the explosion. The alpha and beta particles have a range of only a few feet and may be neglected. Neutrons, which have a range of about 700 yards, are not likely to be a serious independent hazard, since anyone within that distance of an air burst would almost certainly be killed very quickly by blast, heat, or gamma radiation. We are left with gamma rays, which have an indefinite but very long range in air. In Japan some people were killed by them nearly a mile away from the burst.

Casualties produced by the air burst can be grouped under three headings:

1. Mechanical, due to blast, wounding by secondary missiles, and crushing by falling debris.

2. Thermal: flash burns, scorch burns and burns due to secondary fires.

3. Radiation:

(*a*) Produced by neutrons and gamma rays from the bomb.

(*b*) By radiation from radioactive substances.

1. *Mechanical.* The enormous pressure at the centre of the burst is dissipated as a blast wave. This pressure wave differs from that produced by an H.E. bomb. The blast wave from the latter passes in a few milliseconds and is succeeded by a period of feebler but more prolonged negative pressure during which most of the damage is done. Buildings tend to fall inwards towards the explosion. The blast from an atomic bomb takes a second or so to pass, and buildings are pushed outwards away from the explosion.

The secondary effects of blast, such as the collapse of buildings, flying debris and glass, are likely to be the most serious casualty-producing factors, resulting in fractures, lacerations and crush injuries. The fractures and contusions should not present any unusual features, but lacerations from flying glass may be more troublesome. In Japan they were often multiple with embedding of small fragments which were difficult to remove. Where radiation sickness developed slight wounds were said to break down and there was an increased tendency to gangrene.

2. *Thermal.* Two types of burns were observed at Hiroshima and Nagasaki. These were generally differentiated as (*a*) fire and flame burns, and (*b*) flash burns due to thermal radiations. Because of the numerous and widespread fires that develop after an atomic explosion, the total number of casualties, fatal and otherwise, due to flame burns will be very large. An atomic bomb releases roughly one-third of its total energy in the form of thermal radiation. For the " nominal " atomic bomb of the type we are considering, the energy emitted in this manner would be about $6{\cdot}7 \times 10^{12}$ calories, which is equivalent to about eight million kilowatt hours. It is evident that this enormous amount of energy, although acting only for a short time (about three seconds), would be expected to produce considerable damage.

The heat flash is capable of directly igniting inflammable material such as dark cloth, paper and dry wood, thereby starting many fires simultaneously over a wide area—under favourable atmospheric conditions up to a distance of two miles from the explosion centre. Clothing, particularly when white or light in colour, does afford protection against thermal radiations, and the majority of flash burns will be restricted to exposed parts of the body. A very high surface temperature is produced for a short time, and within the depths to which the thermal radiations penetrate the tissues appear to be completely destroyed. Marked redness of the skin appears almost immediately, with progressive darkening and blistering taking place over a period of a few hours. Uninfected first degree burns that are not irritated heal promptly, and keloid formation is not a special feature of the flash burn. For practical purposes of diagnosis and treatment it is not necessary to distinguish between burns caused by flame or by thermal radiation. In British cities two factors are expected to reduce the incidence of flash burns among people in the open. The weather is seldom clear over a city, and even a moderate amount of haze or mist causes a substantial reduction in the range of the thermal radiations. The second factor is that buildings provide a good measure of shielding, particularly in the centre of a city where streets are narrow and buildings high.

3. *Nuclear Radiation.* The injurious effects of radiation from the explosion of an atomic bomb represent an aspect completely absent from the conventional bomb burst. It should be clearly understood, however, that this does not imply that radiation is the most important source of casualties in an atomic explosion. In Japan, a maximum of 15 per cent. of the fatalities were attributed to nuclear radiation, compared with over 50 per cent. due to flame burns and blast, and more than 20 per cent. due to heat flash.

The effects of ionizing radiation on living organisms depend not only on the total amount absorbed, but also on the rate of absorption, and on the area of the body exposed. As far as the effects of an atomic bomb are concerned, the situation is simplified by the fact that the initial nuclear radiations are emitted for a short period so that exposure may be regarded as being of the

acute type. On the other hand, residual radiations, due to fission products contaminating soil or materials, would represent a chronic hazard. Most of the victims of initial nuclear radiations in Japan were exposed over a large part of their bodies, since clothes are no protection against gamma rays. The cases reported have been roughly divided into three groups:

Those receiving:

(*a*) A lethal dose, *i.e.* 600r or more, which is fatal in nearly all cases within two weeks.

(*b*) A median lethal dose, *i.e.* about 400-500r, resulting in death to 50 per cent. in from two to twelve weeks.

(*c*) A moderate or sublethal dose, *i.e.* 100-300r which is generally not fatal.

The Acute Radiation Syndrome

Following exposure to a dose of several thousand roentgens death may occur in a few hours, but there are few reliable observations on such cases. In cases of exposure to lethal but not extreme doses, varying degrees of shock appear within a few hours accompanied or followed by nausea and vomiting. Diarrhoea and fever of stepladder character begin during the first one or two days, with almost complete disappearance of the leucocytes. Scattered petechiae, and increasingly severe diarrhoea progress to sudden death in from four to ten days after exposure.

After exposure to a median lethal dose, nausea, vomiting, loss of appetite and malaise appear within a few hours. After the first day or two, all symptoms disappear and a latent period occurs, lasting up to two weeks, during which time the patient feels relatively well. During the third week there is a recurrence of symptoms which increase in severity with stepladder fever, bloody diarrhoea, loss of hair and ulceration about the lips extending throughout the alimentary tract in the terminal stages.

There is an immediate lymphopenia, followed by marked depression of the levels of all the white cell elements, due to destruction of bone marrow and lymphatic tissues. There is also a fall in the red corpuscle and platelet count. The blood

count reaches its lowest level after about a week and then, in patients who are recovering, begins to increase. Where the symptoms are most severe death occurs within three to twelve weeks after exposure. Those who survive for three to four months and do not succumb to lung diseases or other complications gradually recover.

After exposure to a sublethal dose, there may be a latent symptom-free period up to two weeks or more: when the usual symptoms of anorexia, malaise, loss of hair, diarrhoea and tendency to bleed appear, but are not very severe. Typical changes in the blood picture are found, but their severity and persistence is much less marked. Without complications recovery is uneventful.

Prognosis

It is clear that cases in Group 2 are those in which expert treatment is likely to be the deciding factor between death and recovery. Differentiation between the three groups at an early hour after admission to hospital is most important.

1. The personal dosimeter if carried will give valuable information, or failing this, the reading of an area dosimeter sent in with the casualty will help.

2. Blood examination. In the early stages the lymphocyte count is the most reliable guide. If the lymphocytes are below 200 per c.cm. after twenty-four hours the prognosis is probably hopeless. If above 1,000 per c.cm. after three days then the injury is probably trivial. Borderline cases may be expected to give counts between 300 and 500 per c.cm. after two days. At the end of the first week the total white count will be valuable. Experience has shown that those with fewer than 800 W.B.C. per c.cm. usually die, and those with more than 1,500 W.B.C. per c.cm. usually recover.

3. Fever is to be expected at some time, but a step-ladder climb in the first five days is of grave significance.

4. Gastrointestinal symptoms. Nausea and vomiting occur early as symptoms of both lethal and median dosage. Vomiting and bloody diarrhoea in the first three days is a sign that more than a median lethal dose has been received, and the prognosis

is correspondingly grave. Where the casualty has received a median lethal dose or slightly less, the nausea, vomiting and malaise are less severe, and usually cease after twenty-four to forty-eight hours. Where the blood picture does not agree with the symptoms of fever and gastrointestinal disturbance, other causes, such as influenza or paratyphoid fever must be excluded.

5. Epilation: early and complete epilation is a serious sign, but not necessarily of fatal significance.

Treatment of Radiation Casualties

Treatment resolves in the main to the group of casualties exposed to a median lethal dose (400—500r) of radiation and can be considered in three phases.

First Phase. During the first few days the patient may be regarded as being in a state of shock, requiring routine treatment with complete physical and mental rest, and conservation of body heat. Water balance must be watched, with intake and output records, and parenteral fluid given if there is evidence of dehydration. Care must be taken in forcing fluid or food by mouth because of local damage to the gut. Appetite is likely to return, and a low-residue high-protein diet, rich in vitamins, is indicated. Owing to the necrosis of lymphatic and haemopoietic tissues there may be a release of histamine-like substances: to counteract the effect of these antihistamine drugs are worth trying. Suprarenal cortical extract, given in 5 mgm. doses at eight-hour intervals, has been reported to be of some value.

Intermediate Phase. This is the period of negative symptoms, but positive signs, and the watchword during this phase is prophylaxis against infection. Good nursing is of paramount importance, for the prevention of pressure sores, and the maintenance of a clean mouth. The sulphonamide drugs are contraindicated. Prophylactic penicillin and streptomycin are good, but necessitate needle injection which in these cases is a potential source of skin infection. Aureomycin, which is effective against a wide range of bacteria and is given by mouth, is the antibiotic of choice. Blood, plasma, and plasma substitutes are not indicated at this stage. It is true that leucopenia exists, but whole

blood transfusion is not the answer. The normal life of the leucocyte is only a matter of a day or so, so that the therapeutic effect, if any, would be transient. These therapeutic agents are likely to be in short supply, and are better reserved for normal traumatic casualties, where they are of known and proven value.

Delayed Phase. The earlier this phase appears, the poorer the prognosis. Death appears to be due either to haemorrhage or infection, but biochemical factors may also play a part. This is the time, if at all, for blood transfusion. Marrow failure means anaemia, and here it may be precipitated by haemorrhage due to thrombopenia and capillary damage. Stored blood will not provide platelets, but fresh blood, if available, may have a positive antihaemorrhagic action.

Toluidin blue, and protamine, given intravenously every other day while bleeding lasts, are said to be of some help. The flavonal glucoside Rutin, which is said to diminish capillary permeability, may be of value. Pentose nucleotide, leucocyte concentrates and marrow transfusions have proved disappointing. Plasma transfusion may help to supply protein for nutritional purposes. The plasma substitute dextran, a complex polysaccharide, which is utilized in the tissues, appears to promise valuable help in emergency.

REITER'S SYNDROME

BY

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Reiter's syndrome comprises:

1. *An abacterial urethral discharge*, frequently associated with dilatation of the renal pelves, less commonly pyelitis, haematuria, prostatitis;

2. *Ocular symptoms*: commonly bilateral conjunctivitis, less frequently episcleritis, iritis or herpes.

3. *Polyarthritis:* and

4. *Muco-cutaneous manifestations:*—hyperkeratosis, balanitis circinata, and circular areas of desquamation on the dorsum of the tongue. There is frequently associated an irregular low-grade pyrexia.

Aetiology

The aetiology is still in doubt. Reiter in his original case isolated a spirochaete, which he termed S. forans, from the blood stream, and named the disease spirochaetosis arthritica. Subsequent workers have failed to confirm this finding. A diversity of organisms, staphylococci, enterococci, diphtheroids, B. coli, etc., have been isolated by various observers from the urethral discharge. But when meticulous precautions are taken to cleanse the meatus before obtaining the specimens no organisms are found in smears or cultures. These organisms must therefore be regarded as contaminants. Pleuropneumonia-like organisms have been suggested as the possible cause by Smith (1942) and Dienes and Smith (1942) who isolated " L " organisms from the urethral discharge in Reiter's disease, while in 1948 Dienes *et al.* demonstrated this organism in the synovial fluid of two cases, a finding confirmed by Warthin in another patient. Harkness (1944) suggested the possibility of a virus—as evidenced by the finding of inclusion bodies in scrapings from

the urethra, conjunctiva, and skin lesions: but later Harkness and Henderson-Begg (1948) isolated " L " organisms in 17 per cent. of forty-one cases of Reiter's disease; at the same time Giemsa-stained smears showed markedly pleomorphic inclusion bodies in the epithelial cells. These cytoplasmic inclusions are compatible with either a virus or a pleuropneumonic infection. Dunham *et al.* (1947) cultivated by egg-allantoic inoculation a filterable agent from the urethral and conjunctival secretion of a case of Reiter's disease. This culture proved pathogenic for mice, 60 per cent. of those inoculated intraperitoneally developing conjunctivitis. Allergy has been suggested by Thiers and Joly (1948) on the basis of an exacerbation of symptoms and fever following the intradermal injections of a small amount of the urethral discharge. While the aetiology still remains uncertain, the presence of " L " organisms in a high percentage of cases of Reiter's disease, and their disappearance following successful treatment suggest that this is the probable causal agent.

Transmission of Infection and Sexual Incidence

The term Reiter's disease is used indifferently to describe the urethritis-arthritis-conjunctivitis syndrome complicating acute, or following upon apparently cured, dysentery; as well as the essentially similar condition following sexual exposure and abacterial urethritis. There is an extensive literature on both the dysenteric and sexual forms, and apart from the urethritis, which may be little marked in the former group of cases, the systemic sequelae follow the same pattern. It is reasonable to assume therefore that the causal agent can obtain entry into the body by two separate portals, the ulcerated large intestine or the inflamed urethra.

The disease is more prevalent in males. In the "dysenteric" group Paronen (1948) recorded thirty-four infections in females in a total of 344 cases. In the "urethral" group Harkness (1945) could only find nine cases recorded in the literature. The author has only recognized four undoubted cases in females in a series of ninety-five. It is possible that many further cases would be recognized if a search for pleuropneumonic organisms and cytological inclusions were carried out in cases of

nongonococcal urethritis, cervicitis, etc., associated with a history of transient conjunctivitis, arthritis, etc.

All the cases recorded in childhood have been in association with dysentery. Corner (1950) reporting one case could only find three other cases under twelve years of age, reported in detail in the literature.

Clinical Course and Symptomatology

The author's experience has been confined to the sexual group of infections. The frequency of involvement of the various systems was:

Sex	Urethritis, Prostatitis, cervicitis, etc.	Conjunctivitis or other ocular signs	Arthralgia, Arthritis, etc.	Keratoderma, etc.
Male 91	91 (100)	60 (66)	71 (78)	28 (31)
Female 4	4 (100)	4 (100)	3 (75)	1 (25)

Percentages in parentheses.

The clinical course may be extremely variable: there are undoubtedly many mild forms, self-limiting or responding to minimal treatment. Approximately 25 per cent. follow a more severe course, lasting from seven to eight weeks up to three to six months. Less frequently there is a series of major or minor relapses: in these cases, examination of the wife (or consort) has shown nothing beyond a mild cervicitis. That reinfection was a possible explanation of the relapses is supported by freedom from recurrence after treatment of the spouse.

Urinary Tract Manifestations. The first symptom usually noted is urethritis which becomes apparent after a long incubation period of from ten to thirty days. In the majority of cases the urethritis is mild, a clear, tenacious urethral discharge being associated with only scanty mucoid urinary threads. There is no accompanying dysuria or increased frequency of micturition and the prostate and vesicles are frequently normal, although 25 per cent. of patients show a symptomless prostatitis. In the remainder of cases there is considerable variation up to the

acute type, characterized by marked dysuria, increased diurnal and nocturnal frequency of urination, and a profuse mucopurulent or purulent urethral discharge, occasionally sanious. There is a marked urinary haze; the prostate and seminal vesicles show marked involvement; there is frequently a generalized cystitis; asymptomatic dilatation of the renal pelves is common in this group, while pyelitis, hydronephrosis, and prostatic abscess have been described. There seems to be little relation between the severity of the genito-urinary symptoms and the subsequent systemic sequelae.

Ocular manifestations. Conjunctivitis, commonly bilateral, is usually the first sign of generalization, and occurs from one to thirty days after the urethral discharge has been noted. It may be transient, mild and persistent, or severe. Episcleritis is a frequent accompaniment while iritis is found in some 5 per cent. of cases. In cases following a relapsing course, there are frequently recurrences of conjunctivitis or iritis. Conjunctival and corneal herpes have been described.

Joint Manifestation. Arthralgia, which frequently precedes the onset of a defined arthritis, may be noted at the time of occurrence of urethral discharge. The pain varies greatly in intensity, is frequently migratory, and may pass off spontaneously, or may become fixed on the development of arthritis. The joints commonly implicated are the same as in arthritis, namely, in descending frequency, the knee, the ankle, the smaller joints of the feet and hands, the elbows, etc. No joint is immune.

In over 80 per cent. of cases the arthritis is acute and polyarthritic and characterized by a rapid effusion into the joint, tense synovial thickening, and reddening and glazing of the overlying skin. Pain is intense. Spontaneous retrogression of the arthritis occurs in from four to six weeks, but this improvement is frequently accompanied by involvement of further groups of joints. Subacute arthritis occurs in approximately 20 per cent. of cases: it may also follow an acute phase or be associated with recurrent attacks of the disease. Pain and effusion are less, but synovial and periarticular thickening gives rise to spindle-shaped swellings. These are well seen in

the metacarpo-phalangeal and inter-phalangeal joints. In the later stages, X-ray examination often reveals areas of osteoporosis towards the articular ends of the involved bones. Periosteal reaction is more rare.

Mucocutaneous manifestations. Balanitis circinata is possibly the most frequent of the mucocutaneous manifestations, is more common in the uncircumcised, and affects the coronal sulcus and the corona glandis. The appearance varies according to the stage at which it is seen. At first there is a heaping up of layers of scales, in circular areas of from 2 to 10 mm. in diameter: later those are shed, leaving smooth, weeping, circular patches on which fresh scales are laid down. Coalescence of the lesions gives rise to linear or serpigenous configurations. Typical hard keratodermic nodules also occur. On the dorsum of the tongue similar patches of desquamation up to 12 mm. in diameter are found.

Hyperkeratotic manifestations (keratoderma blenorrhagica) are most common on the soles of the feet and on the glans penis, but may occur anywhere on the body. The sequence is first a vesicular eruption which passes into a pustular stage: this is followed by progressive keratinisation in the walls of the pustule. The core becomes dried up, waxy, and cornified. The resulting lesion is raised, limpet-shell-like, hard, and has been aptly compared to "mountains on a relief map". The hyperkeratotic nodules may remain discrete or coalesce to form plaques of varying extent. Soft parakeratotic lesions may also occur, especially on the scalp, consisting of many layers of scales, often raised in the centre, and without cornification or keratinisation. Balanitis circinata is essentially a soft parakeratosis, and the tongue manifestations also fall into this category.

Pyrexia. Some degree of fever is found in 80 to 90 per cent. of cases during the period of systemic involvement. The temperature is irregular and swinging up to 100 to 103 deg. F., and is associated with marked cachexia and wasting of the muscles controlling the involved joints. The erythrocyte sedimentation rate is raised in proportion to the severity of the disease. Despite the fever and cachexia, the appetite is unimpaired.

Diagnosis

Clinically, the complete syndrome presents no difficulties. In incomplete syndromes, how many of the metastatic sequelae are required to complete the diagnosis? Keratoderma alone is sufficient, otherwise the combination of urethritis, ocular, and joint manifestations or balanitis is essential. In many minor cases careful questioning will elicit the transient occurrence of arthralgia or conjunctivitis, while careful examination will reveal residual joint or dermal lesions. Nevertheless, many cases of non-specific urethritis, in which it is impossible at first to make a diagnosis, ultimately prove to be Reiter's disease.

Clinically the disease may be confused with acute rheumatism, metastatic arthritis in association with blood-borne infection, true gonococcal arthritis, etc., while the occurrence of keratoderma may suggest arthropathic psoriasis. The character of the joint fluid, straw coloured, turbid with a cellular content of polymorphonuclear leucocytes and lymhpocytes, and bacteriologically sterile, is of help; while the demonstration of pleuropneumonic organisms or inclusion bodies in the various secretions is confirmatory.

Treatment

Local measures, urethro-vesical irrigation with 1:10,000 solution of oxycyanide of mercury, prostato-vesicular massage, etc., aid in the eradication of the genito-urinary focus. Parenteral administration of calcium salts results in temporary symptomatic relief of the conjunctivitis, and appears to limit the extent of joint effusion. Streptomycin, 4·0 g. daily for seven days is successful in a number of cases: chloromycetin, in a dosage of 0·5 g. two-hourly for six doses, then four-hourly for the next three to four days, has proved even more effective in a short series of cases. Aureomycin is reported to show equally promising results. A number of the chronic relapsing cases react well to gold salts, *e.g.* myocrysin in a dosage of 0·2 g. in five weeks.

The most uniformly successful method of treatment is pyrexial, preferably carried out in a fever cabinet, the temperature being raised to 105 to 106 deg. F., and maintained for

from five to eight hours. One to three exposures are adequate: relief of arthritis is spectacular, and is paralleled by the improvement in the eye symptoms. The progress of keratoderma is cut short. The alternative to cabinet-hyperpyrexia is the induction of a series of fevers by the intravenous injection of T.A.B., the commencing dose being 25 million organisms. A temperature of 102 to 104 deg. F. is reached and persists for from two to five hours. Six to twelve fevers may be required, at two to three day intervals. Physiotherapy is required to aid restoration of full joint movement, and recovery of the wasted muscles.

Male, M.8426, *aet.* twenty-five, married.

1943–44	Non-specific urethritis—treated in the Navy with sulphonamides and irrigations—outcome satisfactory.
1949	
January	Non-specific urethritis, slight urinary haze, no prostato-vesiculitis or other complications.
March	Hyperkeratotic balano-posthitis, in coronal sulcus. Condition clear April, 1949—treated sulphathiazole and irrigations.
July	Recurrence of non-specific urethritis—clear in one month.
December	Slight recurrence of discharge—clear in one month.

In the various examinations, the condition was very similar—scanty mucoid to mucopurulent urethral discharge—slight urinary haze or threads—no prostato-vesiculitis. W.R. and Kahn consistently negative. Smears, pus ±, no gonococci: cultures, no gonococci, no predominant organism.

1950	
30th August	Recurrence: slight mucopurulent urethral discharge—urine clear—very few threads—prostate and vesicles normal.
3rd September	Arthralgia R. knee, conjunctivitis and photophobia.
6th September	Acute hydrarthrosis R. knee—synovial thickening.

Over a period of the next six or eight weeks the following joints were involved in sequence, the picture being the same in each case, namely hydrarthrosis with marked synovial thickening, L. knee, R. and L. ankles, R. wrist, L. wrist, carpal and interphalangeal joints of R. hand and then L. hand.

18th September Balanitis circinata.

25th October Keratodermic lesions, soles of feet, under toe nails, and distal third of leg.

X-ray: joints showed no bone changes.

The erythrocyte sedimentation rate was greatly increased.

Throughout the infection there was a slight, irregular, low-grade fever.

Treatment

7.9.50—13.9.50 *Streptomycin* 4·0 g. daily (total 24·0 g.). Ineffective.

29.9.50—5.10.50 Distaquaine 0·3 mega unit daily (total 2·1 mega units). Ineffective.

4.10.50—6.11.50 Intravenous T.A.B. fevers, dosage 12·5 to 100 million organisms.

Gradual improvement, paralleled by return of erythrocyte sedimentation rate to normal. The residual fusiform thickenings of interphalangeal joints resolved completely under short-wave therapy. Now completely fit and is doing full-time work as a general labourer.

REFERENCES

1. Corner, B. D. (1950). *Arch. Dis. Child.*, *25*, 398.
2. Dienes, L., Ropes, M. W., Smith, W. E., Madoff, S., and Bauer, W. (1948). *New Engl. J. Med.*, *238*, 509, 563.
3. Dienes, L., and Smith, W. E. (1942). *Proc. Soc. Exp. Biol..*, N.Y., *50*, 99.
4. Dunham, J., Rook, J., and Belt, E. (1947). *J. Urol.*, *58*, 212.
5. Harkness, A. H. (1945). *Brit. J. Ven. Dis.*, *20*, 93.
6. Harkness, A. H., and Henderson-Begg, A. (1948). *Brit. J. Ven. Dis.*, *24*, 50.
7. Paronen, I. (1948). *Acta Med. Scand.*, suppl., 212.
8. Reiter, H. (1916). *Dtsch. Med. Wschr.*, *42*, 1535.
9. Smith, W. E. (1942). *J. Bact.*, *43*, 83.
10. Thiers, H., and Joly, L. (1948). *Munch. Med. Wschr.*, *68*, 769.

BACKACHE, SCIATICA AND RUPTURED DISCS*

BY

KENNETH PRIDIE, F.R.C.S.

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Mr. Pridie prefaced his address by saying that his title would appear to cover a comparatively wide field. Yet what he really proposed was to discuss the relationship of disc disturbances to backache and sciatica, and to demonstrate that certain disc disturbances give rise to a definite clinical picture. Showing a number of slides and drawings explaining the development and anatomical position of the nucleus pulposus and its surrounding structures, he demonstrated that sudden and unexpected or unusual forces will cause weakness or rupture of the annulus fibrosus, and the consequent protusion of the nucleus through the affected part of the annulus. This occurs mainly posteriorly and just to the right or left of the midline. Any degree of protusion may occur, from a mild bulge to almost complete extrusion of the nucleus. This leads to pressure on the dura itself or on the nerve roots as they leave the dura. The signs and symptoms of this condition are reasonably clear-cut. The patient has the back locked in a position of flexion or extension: and when examined in an upright posture and asked to bend forward, when the back is fixed in extension, no flexion takes place in the lumbar region. The lumbar region is maintained stiff and he bends to the right or left, causing a scoliosis away from the side of the affection (this point was illustrated by lantern slides and a most instructive film). The condition may

* An address to the Torquay and District Medical Society at the Torbay Hospital, on Thursday, 25th January, 1951.

be confirmed by mapping out in the affected leg definite areas of altered sensation (paraesthesia), depending on the segmental nerves involved. There is nearly always a history of unwonted or unusual exercise or a twist or strain, which was followed by acute pain in the back, and is associated with sciatica which may come on immediately or at a later date.

For the acute cases treatment consists first of rest in bed for a month followed by the prohibition of heavy lifting or hard work for a further month. If this fails, then more rigid immobilization becomes essential and the patient should be immobilized in a plaster jacket for at least a month or six weeks. If then the patient is no better operation should be considered. Chronic cases or cases seen late are best treated by manipulation. It is preferable to do this without an anaesthetic and the manipulation should always be in a position of flexion. In some cases this will give immediate relief as it relieves the pressure on the nerve roots.

Skiagrams should always be taken when the condition is one of slowly progressive pain, rather than an acute sudden attack coming on after an accident, in order to eliminate any other pathological changes in the bone. It was emphasized that the osteophytic spurs seen around the edge of the vertebrae were indications of old lesions and often had no connection with the condition of which the patient is complaining. Some indirect evidence, however, may be obtained by noticing the narrowing of the intervertebral joint spaces posteriorly, particularly between Lumbar 5 and Sacral 1. A dead straight back in an X-ray, that is, complete loss of the lumbar curve, is an indication of a disc lesion.

The contra-indications to manipulation are pathological changes showing in an X-ray film, slow development of the pain and deformity or if straight-leg-raising is markedly diminished. If manipulation is undertaken in these cases further disc protrusion may be caused. Manipulation should also not be undertaken where there are marked neurological signs, such as loss of ankle or knee reflexes, weakness of the extensors, peronei muscles or quadriceps. The indications for manipulation would be that the patient has had relief from this method of treatment before, or a chronic case where the clinical signs are not very marked and where there is no sign of a tension lesion.

If all the above methods of treatment fail then operation should be considered and the indications for this are:

(*a*) Failure of conservative treatment to relieve pain.

(*b*) Severe and persistent pain with definite physical signs.

(*c*) Well-marked neurological signs; particularly diminished straight-leg-raising, diminished or lost ankle jerks, and weakness of muscles. It has been observed that a completely lost ankle jerk is seldom recovered whereas a diminished ankle jerk will return to normal in about a year.

Editorial

Our Function

Nearly twenty years ago* the functions of the Provincial Medical Journal were thus described: "It gives opportunities for recording cases that would be crowded out of the larger general medical publications, or would be unacceptable to the specialized periodicals. It stimulates medical men of the district to send much that might otherwise be lost: and to prepare their communications with greater precision, since they will afterwards be published. Finally, a local journal forms a mirror of the activities of the medical men of the district, it records passing events. . . ."

As to the last, we can only record those events of which we are cognisant: and are very much dependent upon local correspondents and the secretaries of various medical societies, having in fact a constant fear of our shortcomings in this respect. In recording the proceedings at local meetings, we are much at the mercy of authors. It is manifestly absurd for a quarterly journal to reprint a communication already published several weeks before.

As far as concerns the Bristol Medico-Chirurgical Society, the *Journal* has the RIGHT to publish the proceedings†. But there is a growing tendency for those who address the Society to ignore that right: and, curiously, this is specially noticeable in the case of Distinguished Visitors, the very persons whose addresses we most wish to place on record. It may be, of course, at times that the subject is not really suitable for discussion at a meeting attended mainly by general practitioners. Perchance it is something specially important, deserving a wider publicity than the "Proceedings" of an obscure provincial society: or is reserved for further use on some other occasion. But of late years only too often the impression remains that the Distinguished Visitor just couldn't take the trouble to think out beforehand what he was going to say, far less to write it down! Maybe members are so flattered that these exalted personages deign to address them that they are ready to ignore deficiencies

* Nixon, J. A.—"Provincial Medical Journals", Hist. of Med. Sectn., B.M.A., London, 1932. *Bristol Med. Chir. Journ.*, XLIX, p. 318.

† Unless specially excepted under Rule 19.

—even of manners. If not, it might be well to remind *all* who address the Society that we expect their communications to be available for publication.

* * *

Ita vita est hominum quasi quum ludas tesseris.
Si illud quod est maxume opus jactu non cadit,
Illud quod cecidit forte, id arte ut corrigas.
Terent.

Old Age

Politicians may deplore " an ageing population " as if it were caused by a wicked conspiracy to frustrate " Planning ". Actually, of course, it means only that many more now live out their full span: there are, in this island, above three times as many people over sixty-five as there were fifty years ago. In this we may well feel some professional pride. But pleasing though it is, this has in turn produced problems which we have not yet solved. For example, there is the very greatly increased need for hospital accommodation for these old people: much of our last issue was devoted to methods of using what we have to the best advantage.

But many of the problems are social and political rather than medical. With strictly limited resources, are we to give preference to the child, whose whole life may be changed by timely treatment; to the family breadwinner; or to the old man who has earned care and comfort by a life of toil? These are questions for all alike: certainly not for the medical profession alone. Let Shaw sneer, this is not the *doctor's* dilemma. Premier, poet, prostitute, paranoiac, to the priest each is alike and equally a penitent, to the physician a patient. (Only a politician could have thought out " Positive Health ": the practitioner knows well that his main business is with the care of the sick.*) Pressure on housing may well mean that if the elderly patient enters hospital for necessary treatment, he will lose the one room he can call his own, and his few bits of furniture—and then what happens when he is fit to leave hospital again? Again, the Welfare State seems to be destroying all family and social responsibility: " Oh, NO, doctor! *We* can't look after father now he needs nursing".

There is some evidence that our masters begin to understand that the population changes mean all must work harder or longer. Consultants and civil servants may now work until sixty-five, magnates and magistrates until seventy. Perhaps we

* Luke V, 31.

shall soon be allowed to continue, like Cabinet Ministers, as long as we seem able. It is not only that for many the "Pension" implies a cruel drop in living standards; " Retirement " is, by itself a very deadly disease.* " The first cause, which no man living can avoid, is Old Age: which being of the same quality as melancholy is, must needs cause it. And common experience confirms the truth of it in old persons, especially such as have lived in action all their lives, had great employment, much business and many servants, and leave off *ex abrupto*: they are overcome with melancholy in an instant."† Only a few have had great employment: but many humbler people, who have led busy and (they trust) useful lives, are just as profoundly affected. Others again, for example, have been keen and regular supporters of club or lodge, perhaps have long carried the tedious and obscure burdens of junior office—gradually as the years pass the realisation grows that the expected reward, President, Grand Master, Great Sachem, will never come. Instead, there creeps in the fear of becoming ὁ κατέχων.‡ The tragedies of age are not entirely financial or physical.

* "Among factors influential in causing people to commit suicide are . . . the relation between our present society and old people. . . . Suicide in the last fifty years has increasingly become a disorder of elderly people ". Swinscow, D., Some Suicide Statistics; *B.M.J.*, 1951, i, 1417.

† *Anatomy of Melancholy*, Democritus Junior; I, § 2, ii (5).

‡ " He who now letteth will let until he be taken away." II Thessal. II. 7.

Meetings of Societies

Bristol Medico-Chirurgical Society

The Seventh Meeting of the session was held in the Physics Lecture Theatre, Royal Fort, on Wednesday, 11th April, 1951. Dr. Howard K. Gray, M.D., F.A.C.S., gave an address on **"The Mayo Clinic"**, illustrated by a film. We are not allowed to publish what he said.

The Eighth Meeting of the session was held on Wednesday, 9th May, 1951. Dr. J. Apley and Professor R. Milnes Walker described **"The Diagnosis and Treatment of Congenital Heart Disease"**. We hope to publish their papers in our next issue.

Complaints have been heard that members of the Society do not receive copies of the Balance Sheet before the Annual General Meeting at which they are asked to approve it. So that by the time it appears in print, as part of the proceedings, the following January, it is too late to discuss

details: for example, why the number of subscriptions is less than the number of members, It is therefore hoped to publish the Balance Sheet in future with the October issue before the date of the meeting.

The South-Western Laryngological Association

The Spring Meeting of the Association was held in the South Devon and East Cornwall Hospital, Greenbank Road, Plymouth, on Saturday, 7th April, 1951. It was preceded by a lunch at the Grand Hotel: but this hardly seems an adequate explanation of the difficulty experienced in finding the meeting place. The local members exhibited twenty cases: six cases of malignant disease of upper jaw, treated by operation and irradiation; three cases of frontal osteomyelitis secondary to sinus disease; six cases of laryngeal or pharyngeal cancer recently treated; and two or three other conditions. A lively discussion took place after the clinical meeting. Mr. A. Leigh, F.R.C.S., was elected President for the ensuing session. The next meeting will be in Birmingham on 14th October.

British Medical Association

Bath, Bristol and Somerset Branch

A meeting of this Branch was held at Taunton on 10th May, Dr. Petty, President of the Branch, in the Chair. Mr. Belsey spoke on **"Recent Advances in Thoracic Surgery"**. He described the effect on infection of the lungs of chemotherapy and physiotherapy. Taking empyema as an example, he pointed out that it now often presents as a sterile condition requiring surgery—thoracotomy and excision of the pleura. He said that because of improved dental hygiene and greater care by dentists, lung abscess was becoming rare: segmental removal rather than lobectomy is the modern surgical treatment, and bronchiectasis and compensatory emphysema were avoided by phrenic section. In pulmonary tuberculosis segmental excision had replaced lobectomy, though thoracoplasty was still necessary in some cases. Cancer of the lung has increased, and it is more often recognized by the general practitioner from the clinical signs than by mass radiography. The early symptoms are a non-productive cough and pain in the chest: the X-ray signs of atelectasis and hilal abnormality are important. Treatment is surgical for X-rays are ineffective. Mr. Belsey also stressed the great value of bronchoscopy; changes in the motility of the bronchus are of great significance. Finally, he discussed strictures of the oesophagus, carcinoma and hiatal hernia, demonstrated by excellent X-ray films; answered many questions and encouraged discussion.

A clinical meeting of the Branch was held at Southmead Hospital, Bristol, on Wednesday, 6th June, the President in the Chair: 152 members attended. Over forty very interesting cases were shown, covering a wide range of conditions, by the following members of the hospital staff: Professor Neale, Drs. Beryl Corner, Airth, J. Apley,

Curwen, J. M. Naish, J. MacRae, A. L. Roberts, K. E. Pearson, P. Phillips, R. Warin, Orr Ewing and W. Lewis; Messrs. Brown, G. Scarff, J. A. Pocock, MacPherson, A. Miller and Capper.

Despite a very warm summer evening, there was an excellent attendance and after the meeting very pleasant refreshments were served. Great credit must go to the nursing and catering staff of the hospital, to whom we were indebted for the use of the buildings and particularly to the Out-Patients Sister, Sister Deal, for her organization and handling of the cases.

Bristol Division

The Annual General Meeting of this Division was held in the large theatre of the Wills Physics Department of Bristol University (Royal Fort) on Wednesday, 2nd May. The great importance of this meeting had been impressed on all members and not very many less than 10 per cent. attended. The following officers were elected for 1951–52:

Chairman. Dr. T. Milling; *Chairman-elect*, Mr. R. G. Paul; *Secretaries*, Drs. W. H. Hayes and R. C. Wofinden; *Treasurer*, Dr. C. Dix; *Representatives*, Drs. P. Phillips, A. G. Heron, T. Milling, W. G. Nott, W. Woolley.

The meeting adopted a special vote of thanks to Dr. H. M. Golding for his very valuable services as Secretary during the last ten years.

Local Medical Notes

The XL Long Fox Memorial Lecture will be delivered in the University on 16th October, 1951, by Professor C. F. Powell, F.R.S., Sc.D., Melville Wills Professor of Physics in the University of Bristol. Subject: **"Physical Principles of Radioactivity and the Release of Atomic Energy".**

Appointments

N. F. W. Brueton.—Deputy Director, South-Western Regional Blood Transfusion Service.

G. H. Darke, F.R.C.S.—Consultant Surgeon, South Somerset.

C. C. Jeffery, F.R.C.S.—Consultant Orthopaedic Surgeon, Exeter.

F. R. Hurford, F.R.C.S.—Consultant Surgeon, Lichfield, Sutton, and Tamworth Hospitals.

T. C. H. Davies, F.R.C.S.—Consultant Orthopaedic Surgeon, Newport.

P. Jardine, F.R.C.S., D.O.M.S.—Consultant Ophthalmic Surgeon, Southmead Hospital, Bristol.

John Winter, M.B., M.Rad., D.M.R.D.—Consultant Radiologist, South Somerset Clinical Area.

J. H. Bergin, M.B., B.S., D.M.R.D.—ConsultantRadiologist, Swindon Area.

University of Bristol

Prizes

Foyle.—Miss M. E. Moore.

Edward Fawcett Memorial.—Miss M. E. Moore.

Francis Dudley.—Mr. H. Savery.

Richard Clarke.—Miss J. K. Lloyd.

Degrees and Diplomas

University of Bristol.—D.M.R.D.: Sheila M. Cameron, A. G. M. Davies.

Royal College of Physicians.—F.R.C.P.: Dr. H. D. Davies, Dr. J. E. G. Pearson.

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